PERIOSTITIS.

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BY

N. SENN, M.D.
(Milwaukee, Wisconsin),
Professor of the Principles and Practice of Surgery and Clinical Surgery.

Reported by William Whitford.

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PERIOSTITIS.

GENTLEMEN: We commence, this morning, the subject of the inflammatory diseases of bone; second only in importance to the subject of fractures. It is well, before calling your attention to the pathological questions involved in this class of diseases, to refer very briefly to the peculiarities of bone-structure as compared with those of the soft parts. The periosteum is a fibrous membrane surrounding bone, all the bones being covered by this structure, with the exception of the internal surface of the cranium and the articular surfaces of bone. Fibrous and unyielding in character, it is divided histologically and anatomically, as well as from a pathological stand-point, into two distinct and well-marked layers,—namely, the *external layer*, consisting of a stroma or nidus of connective tissue, with its fibres arranged both parallel and reticular, and the *internal* or *osteogenetic layer*,—the so-called cambium of Billroth,—composed of a layer of cells which we term osteoblasts, and which are attached to a fine reticulum of connective tissue between the external and the internal layer, which also constitutes the portion

of the periosteum destined to furnish material for the growth and regeneration of bone. If we consider the denseness of the structure, its firm attachment to the subjacent bone, and more particularly the anatomical relations existing between the vascular supply of the periosteum and the bone proper, we can readily imagine that any disease affecting primarily the periosteum or the bone is destined to affect the remaining structure by extension of the disease along the fibrous tissue and blood-vessels, from the periosteum to the bone or from bone to periosteum. The older pathologists taught that whenever bone was denuded of its periosteum it was invariably destined to become necrosed, believing that the essential vascular supply was furnished by the periosteum. Clinical observation and experimental research, however, have shown that the vascular supply derived from the periosteum is not essential to the nutrition and growth of the subjacent bone. It has been demonstrated by experiments on animals, taking the case of the shaft of a long bone for instance, that if a ring of periosteum is removed under proper antiseptic precautions, if the bone is fractured at this particular point, after the healing of the external wound, the formation of callus progresses the same as though the periosteum had not been injured or removed, showing conclusively that the callus which is destined to unite the fractured ends of the bone is not derived exclusively from the cam-

bium, but its formation may take place entirely from the fractured surfaces of the bone itself, without participation of the periosteum or surrounding tissues.

Great confusion has existed in classifying inflammatory diseases of bone. At one time pathologists believed that nearly all of the inflammatory diseases originated in the periosteum. It is well to state that, as a primary disease, periostitis is an exceedingly rare affection; as a secondary lesion, it occurs during the course of almost every form of inflammation of the subjacent bone-tissue. Again, it was assumed that inflammation had its starting-point in the bone-substance proper: hence we read and hear so much of osteitis. Later research, however, and the results of experimentation have shown conclusively that ninety-five out of every hundred cases of inflammatory affections of bone originate primarily in the medulla,—that the so-called cases of primary periostitis and osteitis are, in fact, in the majority of cases, only secondary lesions following osteomyelitis. It has been observed in pathological specimens in all stages of inflammation and degeneration that the primary seat is almost invariably found in the medullary tissue; that the bone-tissue proper —the bone-cells—simply occupy a passive *rôle* in the inflammatory process. This is easily understood, if we consider, by analogy, inflammation as it affects the soft tissues in other organs. You will remember that the blood-vessels and connective tissue

are usually the seat of primary changes in all forms of inflammation,—that they constitute the primary seat of degenerative changes and of transformation of mature tissue into inflammatory, embryonal, or granulation tissue. The more essential structures usually are passive in the inflammatory process. The same holds true in cases of inflammation of bone. We can readily conceive, if the periosteum is the primary seat of inflammation in cases of periostitis, that by extension of the process along the blood-vessels and Sharpey's fibres of connective tissue which enter the subjacent bone, forming an uninterrupted vascular net-work and a continuous connective-tissue reticulum between the bone and the periosteum, inflammation can extend from the periosteum into bone without primarily originating in the medullary tissue. It would be more proper to designate such cases as interstitial inflammation of bone, for the same reason that we speak of interstitial inflammation of the kidney, liver, and lungs. The primary pathological changes take place in the blood-vessels and in the connective tissue around the vessels, while the essential histological elements occupy a passive position, suffering secondarily from nutritive changes due to pressure, from the inflammation in exudation, which causes degeneration and absorption of the tissue-elements when the process is slow, and death or necrosis when the exudation takes place rapidly. Consequently, in our classification of in-

flammatory diseases of bone, I will call your attention (*a*) to periostitis as a primary disease, premising that as such it is an exceedingly rare affection.

I will first discuss its ætiology. In cases of severe contusion, of compound and subcutaneous fractures, affecting primarily and directly the periosteum, the injury being followed by a certain degree of inflammation, of a productive character, results in the exudation of formative material underneath the periosteum, the inflammatory process results in infiltration of the connective tissue by migration of white corpuscles underneath and into the interstitial meshes of the periosteum. We have, then, a condition of subperiosteal infiltration; in fact, all changes of a histological character destined to form tissue of its own kind, which serve as a basis for the production and reparation of the broken ends of the bone. Consequently, traumatic periostitis is invariably of a productive character, provided, however, that no infection has taken place. The process is one of restoration of the lost relations of the injured parts. For instance, if the periosteum has become separated over a considerable surface of bone, as we find it frequently in cases of fractured rib, one side of the periosteum being lacerated, the other by a displacement between the ends of the fractured bone becoming separated or detached, with certain spaces between it and the bone, the result is a direct adhesion between the denuded

bone and the separated periosteum through the medium of an exudation which takes place under the cambium from the lacerated vessels and the stable tissue-cells at the site of injury. This form of periostitis you will recognize, clinically, by a slight degree of pain, a moderate amount of swelling, subsiding as the process of organization proceeds and as reconstruction takes place. This is the so-called productive periostitis, as we observe it in cases of injury and disease, uncomplicated by infection. Its infective forms, on the other hand, more particularly that form which is the result of invasion of the specific microbes of suppuration at the seat of contusion, or fracture, or the indirect localization of the microbes through the medium of the circulation, invariably results in a process of destruction (suppuration). From your lectures on "Suppuration" you will remember that we made the positive assertion, "no microbes, no suppuration." This assertion holds true in cases of periostitis. Periostitis not resulting in suppuration is produced by entirely different causes. As a primary disease, suppurative periostitis in its acute form is almost unknown. Secondary suppurative periostitis occurs in the majority of cases as an almost constant result of osteomyelitis, infection taking place by the specific pus microbes, the staphylococcus pyogenes aureus and albus. The extent of the periostitis is indicated by the area of the subjacent primary bone-disease, the periostitis corre-

sponding with the extent of microbic invasion. This form is characterized by a detachment of the periosteum from the subjacent bone, by the subperiosteal suppuration; consequently, in cases of this kind, on exposing the bone you will find the periosteum detached over the same area that marks the extent of the primary bone-disease. We must not necessarily conclude from the existence of periosteal separation that the bone underneath is necrosed. The same reason which explains the integrity of reproduction of bone when deprived of the vascular supply from the periosteum will tend to show that, in pathological conditions, vitality of bone need not be impaired, from the fact that the periosteal blood-supply has been sacrificed by the inflammatory process. The bone may still receive an adequate blood-supply from the nutrient artery and the vessels entering the epiphyseal extremities of the long bones. Extension of the process takes place along the vessels, the microbic invasion being carried on through the medium of the vessels connecting the periosteum and the bone. Under favorable circumstances, after an early evacuation of the products of inflammation, the bone not having become necrosed, the abscess cavity assuming a healthy granulating process, the detached periosteum may again become adherent to the apparently-dead bone, and the process of restoration *ad integrum* is completed. If the vessels within the bone become obliterated by

embolism or thrombo-phlebitis, necrosis is an inevitable occurrence, as these pathological conditions, from the very nature of their causation, are necessarily attended or followed by suppuration. You will recognize this form clinically from the intensity of the local and general symptoms and the rapidity with which the process extends. The entire shaft of a long bone may become denuded within forty-eight or seventy-two hours. It can be recognized again by paying proper attention to the clinical history of the case, by tracing the disease to the primary cause, suppurative osteomyelitis. Clinically, this form is important to remember from the fact that the suppuration is prone to extend to proximate joints. The periosteal separation continuing along the epiphyseal line may produce a separation of the ligaments attached to the epiphysis, which may open a direct entrance of the pus microbes into the adjacent joint, thus producing direct invasion of the adjacent joint. The joint may also become affected by the infection gaining access through a perforation of the epiphysis, or by a secondary infective inflammation of the synovial membrane.

Another distinct form (and one that we recognize as usually commencing primarily in the periosteal structure) is the so-called syphilitic periostitis, due to the action of the syphilitic virus directly upon the periosteum. This form usually assumes a productive character; it seldom results in suppuration; it constitutes one of the

various and multiple manifestations of tertiary syphilis. It is one of the more constant and later clinical features of this disease, affecting most frequently the cranium or the anterior surface of the tibia; the swelling usually assumes a circumscribed form, with well-marked margins, the exudation taking place into and underneath the periosteum,—the result of tissue-proliferation from the internal layer or cambium of the periosteum. Instead of the products of proliferation giving rise to the formation of pus, they form embryonal tissue from the mature histological elements of the periosteum, and the process ultimately will terminate in the transformation of granulation-tissue (1) into cartilage and (2) into bone in the formation of so-called syphilitic exostosis. You have learned that in fractures of the skull union takes place by a minimum amount of provisional callus; consequently the product of tissue-proliferation in syphilitic periostitis of the skull is more likely to be a granulation-tissue, and this granulation-tissue, on account of its failure or absence of the osteogenetic influence from the subjacent bone, is destined to undergo degeneration, softening, removal by absorption, or suppuration.

Another and exceedingly important form of periostitis is the so-called tubercular periostitis,—a periostitis due to the presence of a different form of microbes—the bacillus of tuberculosis; consequently, the products of inflammation are varied. It is

a well-recognized fact that this form may exist as a local disease, independently of primary disease, in the lungs, although more frequently it occurs as a secondary lesion. The product of exudation takes place between the vessels and into the connective-tissue spaces, which produces a local anæmia at the site of the exudation; consequently the product does not go on transforming itself into tissue of a higher type, but terminates in fungous granulations and caseation, a conversion of the products of a low type of tissue-proliferation into a cheesy material; this cheesy material acting as an irritant favors invasion of the tissues by the bacillus of tuberculosis, the area of anæmia increases, and we have added to the tubercular degeneration an additional element consisting of inflammation of the underlying bone, attended usually by a secondary form of infection by pus microbes, which determine suppuration. It is recognized clinically by the peculiar form of caries or destruction of bone underneath the periosteum, the granulations permeating and destroying the bone at different points, so that the surface of the bone presents a honeycombed appearance. If you examine an abscess-cavity the result of tubercular periostitis, you will be impressed with the fact that the internal lining of the abscess-walls consists of a deep layer of granulations, which usually have invaded the adjacent tissues. If the abscess has opened spontaneously, you will recognize the char-

acter of the disease by an extensive undermining of the skin and the absence of symptoms indicative of acute inflammation; you will determine the nature of an ulcer thus formed by the characteristic pathological features presented,—the undermined margins, the flabby granulations invading the subcutaneous tissues as well as the superficial portion of bone, the process of destruction advancing as infiltration proceeds.

In making your differential diagnosis between the preceding forms of periostitis and the tubercular variety, you will endeavor to elicit evidence of tuberculosis in other parts of the body by carefully inquiring into the clinical history of the case, and by subjecting the various organs which are the most frequent seat of the tubercular process to a scrutinizing examination. Usually the epiphyseal extremities of long bones and the short bones are anatomically predisposed to localization of tubercular processes.

Some forms of periostitis are diffuse from the very beginning, as the primary cause invades the tissues in rapid succession; for instance, infection by the specific microbes of suppuration, in cases of acute suppurative osteomyelitis, attacks simultaneously an extensive surface of bone and periosteum. On the other hand, the presence of both the syphilitic and tubercular virus usually produces, primarily, a periostitis of a circumscribed character. The acute form of diffuse periostitis being usu-

ally a secondary affection, the primary form, on the other hand, is noted for its tendency to remain local and for its chronicity.

From a pathological stand-point we recognize three distinct and well-marked varieties of primary periostitis,—the osteoplastic, the suppurative, and the fungous. The osteoplastic periostitis usually follows traumatism when no infection has taken place. The suppurative or destructive form is due to the invasion of pus microbes, the presence of which invariably produces suppuration, when the local conditions are favorable, whatever tissues may be involved; the tubercular variety terminates in caseation, suppuration following as a secondary sequence only after the tubercular deposit has become the seat of a secondary infection caused by the presence of pus microbes. The so-called periostitis granulosa, or granuloma of Friedmann, is that form which is destined to remain stationary, which shows no tendency to transformation of the granulations into tissue of a higher type. This form, primary or secondary, as the case may be, either originating in the periosteum itself or as the result of extension of the fungous process in bone, is noted clinically for its chronic character. Instead of an intense pain marking its onset, it comes slowly and insidiously; the tissues not being infiltrated rapidly by the products of inflammation, we have a process which, instead of making painful tension underneath the

periosteum or within the bone, is accompanied *pari passu* with disintegration of pre-existing tissue, consequently the space occupied by the granulations is created at the expense of pre-existing tissues.

Periostitis granulosa is noted for its obstinacy to all kinds of treatment short of a thorough removal and for the latency with which the symptoms appear; consequently its clinical feature is best expressed by the word "progressive." This form is always caused by invasion of tissues by the specific germs of tuberculosis. When suppuration has taken place in the interior of such a swelling, the abscess-cavity contains flocculent pus and the various products of degeneration, while its walls are lined with granulation tissue. While the interior of the abscess contains but few, if any, of the bacilli, the germs are always found present in its walls, as has been observed by Koch and others who have made this subject a special study.

In considering the symptoms and diagnosis of periostitis, I call your attention first to the most conspicuous and important symptom, pain. Periostitis as it affects the distal phalanx—the so-called paronychia (felon)—in the majority of cases is the result of infection due to traumatism; the injury may have been insignificant and on that account may have been lost sight of when the disease manifests itself. In this particular locality, on account of the unyielding structure of the tissues around the periosteum, but more particularly on ac-

count of the rapidity with which the process of exudation takes place, we have presented to us in a well-marked form that important pathological factor in the causation of pain,—tension. As an early diagnostic evidence in periostitis, pain is an important symptom, and is relieved only by removing tension. Tenderness is even a more important diagnostic element than pain itself. While pain may be present in some forms,—by this I mean a spontaneous or idiopathic pain,—you occasionally will find, by testing the effect of pressure, evidences of tenderness before the patient complains of pain, as, for instance, in cases where the disease is deeply located,—where, upon examination, you are not able to recognize the presence of swelling, and where, from the absence of more reliable symptoms, you may have to rely almost exclusively upon this clinical evidence in arriving at a correct diagnosis. Again, we have repeatedly stated that periostitis manifests a decided predilection for the neighborhood of joints; and when the disease is deeply situated,—as, for instance, near the hip-joint,—circumscribed and long-continued tenderness may indicate to you the existence of localized periostitis, in the absence of more decisive symptoms. If the symptoms in such a case remain circumscribed, local, you may properly suspect the existence of periostitis, secondary or primary, as the case may be. The swelling in periostitis is the result of exudation and tissue-prolif-

eration, within and underneath the periosteum, and when the disease has extended beyond its limits in a peripheral direction, the paraosteal tissues are also affected. As long as the exudation remains limited to the space beneath the periosteum, the swelling is slight, as the subperiosteal space is limited; but as soon as the external layer of the periosteum participates in the process, a paraosteal exudation is added, and the rapidity with which the swelling forms increases. You can readily imagine, from the firmness with which the periosteum is attached to bone, that if the product of inflammation is of a plastic character the space for the exudation is circumscribed, consequently the swelling is slight. In chronic cases, therefore, with a tendency to the formation of productive material, the swelling is moderate and increases slowly, while, on the other hand, in acute cases the swelling is slight as long as the products of inflammation and infection are limited to the periosteum, but as soon as the inflammation has extended to the tissues around the periosteum the swelling increases more rapidly in size as the necessary consequence of an increased area of tissue-proliferation. If the periosteum is incised at this stage of the disease, we have the means of direct inspection of the subjacent bone. In cases of acute periostitis you may find the bone denuded over a considerable area, and presenting all the superficial evidences of necrosis; in chronic periostitis we have either the honeycombed

appearance of carious bone characteristic of tuberculosis, or the floor of the abscess is lined by a continuous uninterrupted layer of granulation-tissue. Inspection alone will give you a true picture of the condition of the bone underneath. In this part of the examination you may combine inspection with exploration, to satisfy yourselves of the condition of the bone. Where periostitis or superficial osteomyelitis has resulted in circumscribed necrosis, we have presented the condition known as *caries necrotica;* but if you wish to examine and ascertain the true condition of the bone without making an incision, you will resort to acupuncture with a disinfected straight needle. If the point of the needle comes in contact with firm, unyielding bone, with a smooth surface, you may satisfy yourselves that the bone is either intact or that separation of the periosteum has taken place without necrobiosis. If, however, on introducing the needle you feel a rough, uneven surface, and you can penetrate the bone, you may rest assured that the periostitis has resulted in caries. Impairment of function may be an important element in diagnosis, and you may have to rely upon this evidence almost exclusively if the disease is located so remotely as to preclude the possibility of eliciting more positive symptoms. The effect of the diseased periosteum upon the function of the part will not only satisfy you that you are dealing with an inflammatory condition either in the periosteum or the

superficial portion of the bone, but it will aid you materially in locating the disease, and to a certain degree indicate its extent. If, for instance, we have a periostitis in close proximity to the hip-joint,—if after eliminating all the elements which we generally expect in cases of intra-articular disease as evidenced by a careful examination as to the effect of pressure and motion upon the joint, we have evidences pointing towards a chronic inflammation of the hip-joint; in such a case, impairment of function limited to the area of motion, with loss of functional capacity of the joint, would indicate its existence.

Infiltration is the first point in considering the morbid anatomy of periostitis. In examining post-mortem specimens, you will have in acute cases, before suppuration has taken place, infiltration of white corpuscles into the connective-tissue spaces and underneath the periosteum. If, on the other hand, you examine the pathological conditions later, at a time when the results of a detrimental action of the pus microbes upon the products of exudation and tissue-proliferation have manifested themselves, you will find evidences of suppuration. If the exudation is of a productive type, you will find an increase in the thickness of the osteoplastic layer, a proliferation of osteoklasts in the substance of the periosteum, and, as a direct result of this tissue-proliferation, a transformation of mature into granulation or embryonal tissue. If, on the other hand, the disease has been the result of infection

with the bacillus of tuberculosis, you will have all the evidences distinctive of tubercular process, wherever found, granulations, or, after suppuration has taken place, an abscess-cavity lined with fungous granulations and miliary tubercles. A careful microscopical examination of the inflammatory product will determine the primary cause in certain forms of periostitis. In cases of acute periostitis attended by suppuration, the vascular supply to the periosteum and bone is diminished by the exudation producing compression ; in other cases the circulation is speedily arrested, as a result of the effect of the primary microbic cause of the periostitis upon the vessels themselves, and may produce a thrombo-phlebitis and a successive venous thrombosis, which are important pathological elements in considering the effects of the circulation upon the products of exudation and the vitality of the tissues immediately concerned in the inflammatory process. In the tubercular variety the gradual diminution of the vascularization of the products of inflammation constitutes a most important factor, the existence of local areas of anæmia determining the fate of the inflammatory product, which is gradually transformed into a caseous material. As the production of new bone depends upon the primary cause, this termination can only be expected in productive or osteoplastic periostitis.

The primary formation of a bone always takes place first in the immediate vicinity of blood-vessels, just the same as in the

formation of a callus after fracture. The deposition of bone takes place after rarefying osteitis or inflammatory osteo-porosis. The death of bone following destructive periostitis may either involve a considerable surface of the bone, producing necrosis, or it may be more circumscribed, resulting in caries necrotica, or it takes place by interstitial absorption, which ends in caries.

In considering the treatment of periostitis, I am happy to make an exception to my general rule in regard to the effects of drugs. It is a well-known fact that syphilitic periostitis will yield very readily to the administration of mercury or iodide of potassium. In cases of this kind *never* resort to operative measures before having thoroughly tested the effects of antisyphilitic treatment. The proof of a syphilitic origin of the periostitis will become apparent in many instances if after the administration of large doses of iodide of potassium the symptoms promptly subside. But the salutary effect of iodide of potassium is not limited to syphilitic periostitis, as this drug exerts a special effect upon other forms of inflammation of fibrous tissue, aside from its antisyphilitic effects, as it constitutes one of the most reliable means to promote absorption if administered internally. By large doses I mean from ten to twenty grains three or four times a day. It is not only prompt in neutralizing the effect of the syphilitic virus, but, by hastening absorption, it diminishes tension, and thus relieves pain.

Its administration always produces a salutary effect in cases of plastic periostitis when following other causes than syphilis. If a case prove obstinate, and iodide of potassium fails, put your patient under the influence of mercury by inunction, or give small doses of corrosive sublimate, combined with twenty or thirty grains of iodide of potassium, until you produce its physiological effect or until the active symptoms subside. Equable compression, by giving support to the circulation, by condensing the connective-tissue spaces, favors absorption, and should always be resorted to. Counter-irritation, by applying tincture of iodine or by blistering, should be employed for the same purpose.

If the pain is intense you may divide the dense periosteum by subcutaneous section or incision under antiseptic precautions, and these measures will prove the most reliable means of securing prompt relief.

In all cases of purulent periostitis no time should be lost in evacuating the inflammatory products by free incision, followed by thorough disinfection of the abscess-cavity, drainage, and antiseptic dressing. Tubercular periostitis calls for thorough removal of all infected tissues by *évidement* or the use of the actual cautery.

All these operations upon bone must be done under the strictest antiseptic precautions. Asepsis in these cases is an essential factor in treatment, as it affords the greatest immunity against further destructive processes in the soft tissues and the subjacent bone.